

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

October 3, 1899.

The President, Dr. Frederick Peterson, in the chair.

CEREBELLAR TUMOR.

Dr. W. B. Noyes presented a boy of five years who had been well until last June. At that time he had an obscure illness diagnosticated as an auto-intoxication. He remained unconscious for two days. During July he had been in fair condition. In August he was noticed to stagger when walking. He could move his legs when sitting, but had absolute ataxia. A slightly spastic condition of the legs interfered with properly obtaining the knee-jerk. Ankle-clonus could be occasionally elicited. No sensory disturbances of any sort existed. His mental condition was becoming slowly worse. Examination of the eyes showed papillitis, with hemorrhages over the disk. The pupils were dilated and did not respond to light. He had neither headache nor vomiting. The case was presented as probably one of cerebellar tumor. The speaker said that it was not impossible that the sickness which occurred last June was a meningitis; hydrocephalus was also possible.

Dr. George W. Jacoby said that he had been impressed with the general laxness of the boy and the bending forward of the thorax. This general loss of muscular tone made him believe the diagnosis of cerebellar tumor to be correct.

Dr. Hughes, of St. Louis, said that the case suggested cerebellar tumor, but it seemed to him not improbable that cerebrospinal meningitis had existed, and that the present condition was the result of this meningitis.

Dr. Edward D. Fisher inclined to the view that the case was one of cerebellar tumor, although admitting the force of the statement of the last speaker.

Dr. Noyes thought that the condition of the eyes alone was sufficient to settle the question of the diagnosis. He had been unable to find the report of a case of hydrocephalus producing optic neuritis, although it might cause optic atrophy when very far advanced. The case was evidently progressing rather rapidly now.

BRACHIAL PLEXUS PARALYSIS FROM PRESSURE.

Dr. William M. Leszynsky presented a young woman having a brachial plexus paralysis affecting each arm, the result of pressure. The biceps group was paralyzed; the extensors and the deltoid were extremely weak, and atrophy was observed about the shoulder joint. No sensory symptoms were detected. The faradic irritability was lost in the nerves and muscles. The same condition, although to a less degree, was present on

the right side. The paralysis had existed three months. The etiology in this case was interesting. The woman had been operated upon about three months ago for an abdominal growth. Immediately afterward a paralysis developed, undoubtedly from the pressure exerted for about one hour and a half by an apparatus used for support. Dr. Leszynsky said he had seen similar cases of pressure paralysis, but in this instance the paralysis had been so severe, and had lasted so long, that it brought up the interesting question as to whether or not the surgeon might be liable to be sued for damages. On inquiry he had learned that this form of paralysis was not at all uncommon at the present time in surgical practice, yet it was entirely avoidable. In an article by Dr. Garrigues several cases had been reported in which the paralysis had lasted for a year or more, and had been followed by atrophy. The apparatus referred to usually made pressure between the clavicle and the first rib.

Dr. Fisher said that these cases of pressure paralysis were quite commonly observed after operations, as for example, after operations for appendicitis; but he had never before seen a case in which both sides had been involved. Negligence in attending to the position of the arm during operation was often responsible for such paralysis.

Dr. Fraenkel said that while many cases of post-operative paralysis had been reported, it should be remembered that in some the paralysis had been ascribed to the ether, although in others the influence of pressure had been definitely shown. This made it difficult to hold the surgeon liable in many instances.

Dr. G. W. Jacoby said that the various cases had been recently tabulated and the conclusion reached that all the paralyses could only be explained by pressure. The consensus of opinion at the present time seemed to be that the ether could not give rise to this form of paralysis. Surgical assistants were often very negligent about these matters. For example, he had found an assistant making hypodermic injections of ether and brandy in a case of collapse, directly over the musculospiral nerve, and the ensuing paralysis he would have found difficulty in explaining had he not seen these injections being made. Again, he had known it to be positively asserted that there had been no pressure, and yet he had personally seen the etherizer resting his elbows heavily over the patient's clavicles.

Dr. Frederick Peterson said that he had seen even the peroneal nerve affected in this way after ether narcosis. In every case that he had investigated he had found that there had been a possibility of the paralysis having been produced by pressure. The question was of great importance, for he believed it would be perfectly possible for such a paralyzed patient to collect damages from the surgeon on account of such negligence. Nearly all the pressure paralyses occurring at night developed during alcoholic sleep, and he had come to look upon these as occurring in nerves which had been rendered especially vulnerable by ether or alcohol, or, as suggested by Dr. G. W. Jacoby, the inhalation of carbonic oxide in close sleeping-rooms. In cases of musculospiral palsy the nerve was generally pressed upon when the patient slept with the arm raised, and the hand under the back of the head.

Dr. Hughes said that the paralyses following the comatose state

in epilepsy exhibited local peripheral expressions. Neurologists had been accustomed to consider these as of central origin. His own opinion was that ether was a factor which could not be ignored—that it was a toxic factor. He had not yet been convinced that these cases were exclusively peripheral paralyses, and, owing to this lack of unanimity among neurologists regarding the causation of these cases, their medico-legal aspect assumed grave importance. Many of these patients recovered, as did the patients with epileptic paralyses, and he was inclined to think that the toxic condition of the brain found associated with etherization was an important etiological factor.

Dr. Leszynsky said that he could not agree with the views of the previous speaker regarding the effect of etherization. When a healthy person was etherized it could hardly be said that the toxic condition could so soon be responsible for the paralysis; it was rather a case of extreme pressure made during unconsciousness. In his own mind there was no question that all of these cases were directly due to pressure made during unconsciousness. Ether, like other stimulants, might give rise to cerebral hemorrhage, or induce hemiplegia of the hysterical type. If these cases were due to a lesion in the anterior horn they would not all recover, as occurred invariably in the class of cases under discussion.

THE CENTRAL NERVOUS SYSTEM IN ACUTE MALARIAL INFECTION.

Dr. James Ewing read this paper. He introduced the subject by reporting the three following cases:

Case I.—*Estivo-autumnal malaria*, with marked cerebral symptoms and infection with a single, well-defined group of parasites. The patient was a soldier who had just returned from Cuba. The stupor lasted thirty-six hours prior to death. Two days before death many ring-shaped organisms in the blood had been found. The stupor began twelve hours after sporulation occurred. At the autopsy the viscera showed the lesions of severe malarial infection. The capillaries of the cerebrum, cerebellum, medulla and cervical cord contained a large number of cells harboring parasites. Many capillaries were occluded by thrombi. The ganglion cells showed everywhere a reduction in size, irregularity and splitting or loss of the chromatic bodies. Death was due to the massing of the parasites in the capillaries. The case was a good example of comatose malaria of the cerebral type.

Case II.—This patient had chills on alternate days for several days, and, on admission, had a pulse of 110, and was much prostrated. Marked insomnia and mild delirium existed for awhile; then the delirium became still more marked, and finally coma developed, lasting for some time before death. No evidence that the coma was uremic was obtained. The temperature reached 108° F. shortly before death. On October 12 the blood contained an enormous number of ovoidal, spherical and crescentic bodies. On October 25, or eight hours before death, very few crescents could be seen. None of the younger forms were found. The autopsy showed moderate edema of the brain, the basal vessels normal, and very few parasites present. The deposit of pigment in the brain was slight. Throughout the cortex the cells showed no marked change further than a uniform diminution in the chromatic substance. In many of the pericellular lymphosites were peculiar structures—elongated fibers or rods with tapering ends—of undetermined nature. Similar deposits were found in other cases of malaria, and in one case of tubercular meningitis. The chief feature of

this case was the prolonged delirium and coma, yet the post-mortem examination showed far less change in the kidney than was usually seen in profound malarial infection.

Case III.—This was a case of fatal malaria associated with hemoglobinuria. The man had contracted malaria in Santiago in July, 1898. He was comatose, extremely emaciated, excessively anemic and moderately jaundiced. He was at Camp Wikoff for three days prior to death. The dura and pia were distinctly jaundiced. The cerebrum, cerebellum and medulla showed the usual injection of vessels with blood, but the vast majority of the vessels were free from the parasites. An abundant deposit of hematoidin in the kidneys was interesting.

Commenting upon the foregoing cases, the speaker said that when the brain cortex was markedly brownish it indicated usually the presence of a large number of parasites, but this was not an invariable rule. The majority of cases of comatose malaria did not exhibit the massing of the parasites in the brain. Of eight cases in which he had examined the brain after death in none was there a distinct brownish discoloration of the brain. Hemorrhages had been found in some cases, as had also a moderate degree of edema. Usually the parasites were uniformly distributed in the brain and cord, but a case had been reported in which they were localized in the medulla. The number of these parasites was often enormous, and complete occlusion of the vessels was not at all uncommon. While most of the fixed pigment was found in the endothelium, the parasites were rarely seen in the endothelial cells. It was probably safe to refer the cerebral symptoms observed in these severe cases of malarial infection to the general condition of the obstructed circulation. The ganglion cells, in cases of comatose malaria, had been studied, and the changes found to consist chiefly in the various degrees of chromatolysis. The dendrites were usually involved before the cell body. The ganglion cells appeared to suffer less than in the average case of typhoid fever. The mere presence of the parasites in the tissues seemed to exert no bad influence, except in a mechanical way. In one of the cases reported in the paper the deepening of the coma could be apparently connected with the progressive filling of the capillaries with the parasites, and the formation of thrombi. In the other two cases the parasites were few and the pigment scanty. In both of these cases the malarial infection was exceedingly severe. These cases showed that the coma of malaria was not always referable to the presence of the cerebral parasites. Of the writer's sixty-four cases of malarial coma reported there were five simple tertian infections. In the estivo-autumnal cases, with crescents only in the blood, there were thirty-four instances from the Montauk camp. Coma appeared to be rather frequent in cases showing only crescents in the blood. Crescents did not exhibit such a tendency to unequal

distribution as did the fertile forms. From the condition of the viscera found in the second and third cases reported—and this variety occurred especially in cachectic individuals in whom the disease had existed for some weeks—it seemed probable that the coma was the result of the general and profound malarial infection. The writer did not believe that much importance should be attached at the present time to the condition of the ganglion cells in malaria. When the coma resulted from the massing of the young parasites in the brain, it was generally gradual in onset, and the prognosis was very unfavorable. In some cases the coma developed suddenly, and was more amenable to appropriate treatment. The general clinical character of these cases indicated that an embolic process was concerned in their causation. Of eleven cases of the first class, reported by the writer, ten proved fatal. To summarize: The three anatomical conditions were: 1. The mechanical obstruction of the cerebral capillaries by large numbers of young parasites; 2. An embolic process causing an occlusion of some vessels (usually temporary) by parasites or pigmented leucocytes, and 3. A general toxemia, such as might occur in other infectious diseases, the blood showing few, if any, autumnal rings.

Dr. W. B. Noyes said that two years ago he had seen a case showing coma, stertorous respiration, partial paralysis and irresponsive pupils. The blood taken from the spleen at autopsy showed the presence of pigmented leucocytes, parasites of the estivo-autumnal variety. Sections from every part of the brain, without staining, showed that the walls of the capillaries were infiltrated with pigment. The absence of the parasites and the enormous amount of pigment were especially noteworthy. The usual lesions of pernicious malarial fever were found in the other organs of the body. In this case the diagnosis during life had been cerebral hemorrhage by almost every physician who had seen the case—certainly the clinical appearances were the usual ones found in cerebral hemorrhage.

Dr. Lewis A. Conner thought the jaundice might explain some of the comas that were otherwise inexplicable. It was surprising sometimes how an apparently moderate degree of jaundice might be associated with very marked cerebral symptoms.

Dr. Fraenkel said that he looked upon most of the nervous consequences of malaria as due to toxemia.

Dr. Hughes said that the paper was very satisfactory in that it explained certain old and well-known clinical facts. Williamson, of England, had been one of the first authors to refer to the effect of malaria on the nervous system.

Dr. William Hirsch reported a case of a gentleman sent to him from the South about one year ago, with a diagnosis of spinal tumor. The symptoms had been atrophy of the muscles of the left shoulder girdle, associated with intense pain. The patellar reflexes were exaggerated. After having had the case under observation for some time, and studying the previous history very carefully, he had become convinced that this was not the correct diagnosis. The patient gave a history of having had a somewhat similar attack some time before, which had been recovered from. While under observation the man had developed a typically malarial temperature, and this had been asso-

ciated with splenic enlargement. Examination of the blood at that time had been negative. After about six weeks of anti-malarial treatment recovery had been complete. A very peculiar symptom in this case was a decidedly tender swelling over the sternum. This had disappeared as the patient had improved. The man, against advice, had returned to his home in the South, and when seen again, a few weeks ago, had been in a worse condition than the year previous. The left clavicle then exhibited a tender swelling, like that observed previously over the sternum. There were similar swellings over one ulna and on one portion of the skull. He was now improving rapidly under anti-malarial treatment. The case was reported because of this peculiar affection of the bones.

Dr. Teller, of Memphis, said that he had practised in a district in which malaria was exceedingly common and very malignant. In his neighborhood these peculiar neurotic manifestations of malaria were so common as to make the physicians constantly on the lookout for them. The cases of cerebral congestion were all too common, and very frequently proved fatal. The physicians of his locality looked upon the disease as a profound toxemia.

Dr. Ewing said that he had been especially interested in the remarks of one of the speakers concerning cases exhibiting jaundice. These cases commonly showed at autopsy profound and peculiar changes in the liver. In the case reported by Dr. Hirsch there were certain features that seemed to him to point to syphilitic infection. Dr. Ewing thought that the medical profession was too prone to jump to the conclusion that a disease was malarial because it seemed to respond to the administration of quinine. A very large number of nervous conditions reported, that had been referred to malaria by the older writers, should be excluded at the present time, because they had not been properly authenticated according to modern notions.

222. ON A FORM OF NERVE TERMINATION IN THE CENTRAL NERVOUS SYSTEM, DEMONSTRATED BY METHYLENE BLUE. W. A. Turner and W. Hunter (*Brain*, 22, 1899, p. 123).

The authors used a method adapted from Ehrlich, as follows: Small quantities of a saturated solution of methylene blue were injected subcutaneously at intervals of from fifteen to twenty minutes until the animal died. The central nervous organs were then rapidly removed, sliced into convenient blocks, and placed in a solution of ammonium molybdate for purposes of fixation. After washing, hardening and embedding, the blocks are cut as usual and mounted with a coverglass. A number of nervous structures were examined from a variety of animals, and the results obtained are given in résumé thus:

1. In the majority of cases a cellulipetal fiber may be traced to, and found to break up over, the cell body and base of the protoplasmic processes. In no case has the cellulipetal fiber been found to break up previous to reaching the cell body.
2. This lattice work is most clearly defined over that portion of the cell, namely, the root of the axis cylinder process, which is deficient in the so-called Nissl bodies.
3. It is also clearly seen over cells whose general staining is faint.
4. When the cell is rendered indistinct by changes in the focus, the lattice work is thrown into clearer perspective.
5. As best seen in the nucleus of the trapezium, the ending may be apparent without the cell. The paper is illustrated by some wretched micro-photographs and some schematic pen drawings. JELIFFE.